Current concepts in the use of PRICE for soft tissue injury management

Bleakley, C. M. (2009). Current concepts in the use of PRICE for soft tissue injury management. *Physiotherapy Ireland*, *30*(2), 19-20. http://www.iscp.ie

Current Concepts in the Use of PRICE for Soft Tissue Injury Management

Bleakley CM[1]

[1]Health and Rehabilitation Sciences Research Institute, School of Health Sciences, University of Ulster, Jordanstown, Northern Ireland

Treating soft tissue injuries with protection, rest, ice, compression and elevation has been the mantra of physiotherapists for many years. Commonly shortened by the acronym 'PRICE', this approach is also widely accepted by layman as an essential component of first aid practice. O'Sullivan and Keane's[1] interesting survey of female Gaelic footballers published in the current issue of Physiotherapy Ireland, sought to determine the depth of athletes' knowledge on using PRICE for self-management of soft tissue injuries. Although most of the participants were aware of its basic concept, there were wide variations in practice, particularly regarding the choice of dosage, and the optimal modality combination. Given that none of the athletes included in the survey were medically qualified, we might have anticipated the observed variation in O'Sullivan and Keane's[1] results. What may be more surprising however, is that similar surveys conducted on physiotherapists and other medical practitioners, have also yielded disjointed results.

So why is there such a lack of consensus when it comes to treating acute soft tissue injuries? O'Sullivan and Keane[1] suggest that shortcomings in the evidence base are undoubtedly a factor. Indeed, the majority of clinical studies in this particular area lack internal validity and few have considered or reported adequate details on treatment dose.[2] However, a lack of good quality randomised controlled trials may just the tip of the ice pack (berg)! Delving into the preclinical research in this area also shows that a number of seemingly basic concepts cannot be fully explained.

For many clinicians, their rationale for using modalities such as ice and compression after an injury is simply that it controls the clinical signs of inflammation. Applying a cold compress on an injury that is hot, red and swollen is commonsensical; but it clearly overlooks other potentially important physiological, cellular and molecular events. We use the term inflammation constantly in the clinic when referring to acute injuries however few can define what they mean by it. There is continued confusion as to whether inflammation is a 'bad' process, or, whether it is in fact fundamental for optimal repair.[3] With major advances in our understanding of the inflammatory response in recent years, we can now begin to put these questions into context and consider the clinical implications and pathophysiological rationale for common interventions such as ice.

Usually soft tissue damage relates to an acute mechanical overload and resultant (primary) cell injury. Although we are aware that an inflammatory response then ensues, many may not appreciate the complexity of these molecular and cellular events. For example there is a growing body of evidence to show that the characteristic rapid influx of neutrophils immediately after injury is an event that requires particular attention. Primarily neutrophils remove tissue debris; however certain aspects of their function may also be destructive to healing. This is thought to relate to their production of oxygen free radicals and toxic enzymes. These products, coupled with the typical ischaemic environment associated with an injury site, can cause further (secondary) cell injury. One of the potential benefits of applying ice is that it decreases the risk of secondary cell injury. This concept is supported by findings that cryotherapy decreases the influx of neutrophils in acutely injured rat tissue.[4] Animal studies also show that cooling reduces the metabolic demand of cells around an injury site, allowing them to survive for longer periods in an ischaemic environment.[5] Although these mechanisms of effect are promising, we must consider their clinical context. To adequately lower cellular metabolism in animal models, treatments of between 5 and 6 hours of continuous cooling were required. Compare this to O'Sullivan and Keane's study[1] where the majority of athletes favoured durations of between just 5 and 10 minutes. These small dosages cause little fluctuation to deep tissue temperature and are therefore unlikely to influence cell metabolism. Employing longer treatment durations in the clinic is one answer; however, we must also consider the associated impracticality, and increased risk of side effects such as ice burns.

Quantifying the degree to which our treatment modalities can target key events such as cell metabolism, neutrophil activity and free radical production will be an exciting area for future research. Indeed, the panacea may be to find a method of inhibiting the damaging effects of cells such as neutrophils, whilst maintaining all their benefits.[6] This is certainly a challenging idea, but it is perhaps unrealistic to expect the humble ice pack to have such a precise effect!

For now, it may be more important to consider the magnitude of temperature reductions that we can achieve safely in injured humans. Whilst influencing temperature in deep tissues remains difficult, skin temperatures can be reduced significantly. The magnitude of the reduction depends largely on the mode and duration of treatment, and the presence or absence of a barrier. It is interesting that O'Sullivan and Keane[1] found very few athletes opted to use crushed ice. Paradoxically, this is one of the most effective modes of cooling, and quickly decreases skin temperature to below 15°C. Physiologically, this is an important temperature threshold to reach, as it is associated with a number of important effects including; decreases in nerve conduction velocity, pain threshold[7] and ultimately pain management. In addition, these effects form the basis for interventions such as 'cryokinetics' which use cold induced analgesia to facilitate weight bearing, therapeutic exercise and normal movement strategies after injury. Currently, it is difficult to suggest how well cryokinetics fit with the protection and rest components of PRICE; however it does suggest that ice could still have a role to play beyond the acute phases of injury.

It is clear that physiotherapists cannot recommend an

optimal protocol for ice application beyond conjecture. Similarly, deciding on the most effective compression bandage or quantifying how much rest to advice, is also challenging. O'Sullivan and Keane's[1] study provides further evidence that developing clearer evidence based guidelines for PRICE is an important aim for the future. However, given the complexity of contemporary models of inflammation, we must also consider if is still realistic to produce one set of definitive guidelines to suit every type of soft tissue injury? Furthermore, we must consider that by recommending PRICE, we are recommending a combination of different treatment modalities each with their own unique molecular, cellular, physiological and clinical effects.

Correspondence to: Dr Chris Bleakley.
Email: chrisbleakley@hotmail.com

REFERENCES

1. O'Sullivan K, Keane N. Female Gaelic Football Players' Acute Soft Tissue Injury Management. Physiotherapy Ireland. 2009;30:13-18.
2. Bleakley C, McDonough S, MacAuley D. The use of ice in the treatment of acute soft-tissue injury: a systematic review of randomized controlled trials. Am J Sports Med. 2004;32: 251-261.
3. Scott A, Kahn KM, Roberts CR, Cook JL, Duronio V. What do we mean by the term "inflammation"? A contemporary basic science update for sports medicine. Br J Sports Med. 2004;38: 372-380.
4. Schaser KD, Disch AC, Stover JF, Lauffer A, Bail HJ, Mittlmeier T. Prolonged superficial local cryotherapy attenuates microcirculatory impairment, regional inflammation, and muscle necrosis after closed soft tissue injury in rats. Am J Sports Med. 2007;35: 93-102.
5. Merrick MA, Rankin JM, Andres FA, Hinman CL. A preliminary examination of cryotherapy and secondary injury in skeletal muscle. Med Sci Sports Exerc. 1999;31:1516-21.
6. Toumi H, Best TM. The inflammatory response: friend or foe for muscle injury? Br J Sports Med. 2003;37:284-6.
7. Algafly AA, George KP. The effect of cryotherapy on nerve conduction velocity, pain threshold and pain tolerance. Br J Sports Med. 2007;41:365-369.